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## **POSTTRAUMATIC GROWTH MODERATES THE ASSOCIATION BETWEEN VIOLENT REVICTIMIZATION AND PERSISTING PTSD SYMPTOMS IN VICTIMS OF INTERPERSONAL VIOLENCE: A SIX-MONTH FOLLOW-UP STUDY**

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The current study was developed to investigate whether posttraumatic growth (PTG) moderates the association between violent revictimization and maintenance of posttraumatic stress disorder (PTSD) symptoms in a sample of 202 Dutch victims of interpersonal violence who had filed a claim for state compensation between January 1st and December 31st, 2006. Based on previous research, it was hypothesized that PTG buffers against symptom increase due to revictimization. A six-month prospective study design was employed to enable adjustment for PTSD symptom severity before revictimization. Regression results indicated that symptom severity at initial assessment predicted symptom severity six months later. No main effects were found for revictimization and PTG. An interaction effect was found between revictimization and PTG. Post hoc probing of the interaction suggested that those with low PTG experienced more severe PTSD symptom levels after revictimization compared to those without subsequent victimization experiences. Implications for victim services practices as well as limitations and strengths of the study were discussed.

Research into the psychological aftermath of traumatic events has traditionally focused on their negative mental health consequences. However, in the last decade a paradigm shift has occurred with many studies reporting on their positive sequelae. Despite this

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changed attitude among scientific scholars, only a few studies have prospectively studied positive changes among victims of trauma (e.g., Fredrickson, Tugade, Waugh, & Larkin, 2003). Furthermore, with regard to violent crime, previous studies seem to have limited their scope largely to rather homogeneous samples of victims, particularly those of sexual assault (e.g., Borja, Callahan, & Long, 2006) and domestic violence (e.g., Cobb, Tedeschi, Calhoun, & Cann, 2006). The current study was developed to address these limitations by prospectively investigating change across a wide array of violent crime categories.

Positive change has been observed following varying types of adversity, such as (natural) disaster (e.g., Cryder, Kilmer, Tedeschi, & Calhoun, 2006), life-threatening disease (e.g., Bellizi & Blank, 2006) and war exposure (e.g., Solomon & Dekel, 2007), and has been labelled with a variety of terms, such as posttraumatic growth (e.g., Tedeschi & Calhoun, 1995), adversarial growth (e.g., Linley & Joseph, 2004) or stress-related growth (e.g., Park, Cohen, & Murch, 1996), and thriving (e.g., Carver, 1998), which all refer to a level of functioning superior to that which existed prior to the traumatic event or circumstances (Linley & Joseph, 2004; Tedeschi & Calhoun, 2004; Zoellner & Maercker, 2006). In this paper the term posttraumatic growth (PTG) is preferred, as this seems to be the most commonly used in psychological literature.

Competing theoretical explanations for the association between maladaptation following trauma, particularly posttraumatic stress disorder (PTSD), and PTG are provided throughout the literature. Levine, Laufer, Hamama-Raz, Stein, and Solomon (2008), for example, have attempted to conceptualize the relationship between PTSD and PTG in either of four forms. First, a positive association between PTSD and PTG may be assumed to exist by those who believe that high levels of distress are a prerequisite for PTG to occur (e.g., Tedeschi & Calhoun, 1996, 2004). Second, a negative relation between PTSD and PTG may be assumed by those who consider PTG as an adaptive outcome of successfully coping with trauma and its negative psychological consequences (e.g., Johnson et al., 2007). Third, a lack of interrelatedness between the two may be assumed by those who see PTSD and PTG as two different and independent dimensions (Linley & Joseph, 2004). Finally, several scholars have proposed a curvilinear relation between PTSD and PTG, with the highest levels of PTG experienced by individuals suffering

from intermediate levels of distress (e.g., Butler et al., 2005; Levine et al., 2008).

However, to date, empirical studies have not been able to provide convincing evidence in support of one of these views; according to a recent review by Zoellner and Maercker (2006), most studies have not observed a significant relationship between PTSD and PTG, while correlations ranged from  $r = -.2$  to  $r = .2$  for the few studies that did find an association. The former were often cross-sectional in nature. Studies that had used a prospective design or assessed PTG through administration of standardized measures, such as the Posttraumatic Growth Inventory (PTGI; Tedeschi & Calhoun, 2004) or the Stress-Related Growth Scale (SRGS; Park et al., 1996), appeared to be most likely to establish a positive association between PTSD and PTG.

Several explanations have been suggested for the inconclusive pattern of empirical results on the relationship between PTSD and PTG. According to Helgeson, Reynolds, and Tomich (2006), who conducted an extensive meta-analysis of 87 cross-sectional studies, one important reason for the inconsistency of findings across studies is the unclear nature of the phenomenon they investigated due to a lack of pretrauma assessment of the (psychological) domains on which growth may be assumed to occur; in many of these studies people's reportings of PTG may have reflected either actual changes in their lives or merely perceived alterations, for example resulting from cognitive strategies employed to construe PTG as a way to relieve distress (see also Sumalla, Ochoa, & Blanco, 2009). Against this line of reasoning one might argue that PTG can only be measured during the aftermath of trauma. Due to this characteristic, pre- and posttrauma measurements of growth-related domains may be assumed to have different meanings (e.g., Allison, Locker, & Feine, 1997)—a phenomenon referred to as *gamma change* (Golembiewski, Billingsley, & Yeager, 1976) and which cannot be captured by a prospective design.

In addition, and in line with others (e.g., Hobfoll et al., 2007; Zoellner & Maercker, 2006), the authors argued that previous research has often failed to test for possible moderator effects between PTG and third variables to identify subgroups of victims with varying levels of interconnectedness between PTSD and PTG (Helgeson et al., 2006). Alternatively, levels of PTG may be assumed to act as a moderator in the relationship between a stressor and PTSD (e.g., Siegel & Schrimshaw, 2007). One of the few prospective studies to

test for moderation was performed by McMillen, Smith, and Fisher (1997). They intended to examine the relationship between benefits experienced 4-6 weeks after the traumatic event and later psychological adjustment in victims of three types of disaster (tornado, mass killing, and plane crash survivors). Among other things, they observed a positive relation between severity of exposure, defined in terms of proximity of the experienced event, and level of distress three years post-victimization. By contrast, an inverse relation was observed between severity of exposure and mental health change in terms of PTSD diagnosis for those who had experienced benefits, suggesting an adaptive role of PTG in the aftermath of trauma. Comparable results have been reported by cross-sectional studies measuring PTG many years after traumatization (e.g., Maercker & Herrle, 2003). However, an important limitation of these studies is that they tested the significance of the interaction between severity of trauma exposure and subsequent PTG and therefore have not been able to assess moderation in a methodologically sound manner; after all, that would have required measurement of growth-related domains prior to trauma exposure (e.g., Kraemer, Wilson, Fairburn, & Agras, 2002).

Given the aforementioned, the current study was developed to further clarify the functional significance of PTG in the aftermath of trauma. More specifically, we focused on the possible moderator role of PTG in the association between violent *revictimization* and persistent PTSD symptomatology experienced in response to the original traumatic violent event among victims of rather diverse categories of interpersonal violence.

Revictimization is broadly considered to act as an indicator of *exposure gradient*, which is an alternative measure of exposure severity (Winkel, 2005). Previous research has indicated that victims of violence run an additional risk of being victimized once more compared to nonvictims (e.g., Smith, White, & Holland, 2003; Bensley, Van Eenwyk, & Wynkoop Simmons, 2003). Furthermore, revictimization appears to play a role in the maintenance of PTSD symptoms (e.g., Noll, Horowitz, Bonanno, Trickett, & Putnam, 2003; Schumm, Hobfoll, & Keogh, 2004; Griffing et al., 2006) and thus may prevent resolution of the event that triggered them in the first place.

By adopting a prospective design and inclusion of participants who had been victimized previously, we attempted to partly avoid the methodological issues discussed above; we presumed that the meaning of PTG regarding the initial act of violence would not dif-

fer before and after revictimization, for all participants had been exposed to a traumatic act of violence at least once before. In addition, the assessment of revictimization enabled us to look for an interaction effect between PTG and subsequent violence exposure on persisting PTSD symptom levels, while simultaneously controlling for the effect of symptom levels experienced prior to revictimization. If such an effect would exist, this would provide preliminary support for the protective role of PTG in the face of violent revictimization. Based on the results provided by McMillen and colleagues (1997), we hypothesized that revictimization would be positively related to victims' PTSD-scores at follow-up, while controlling for baseline symptom levels. However, it was additionally expected that high PTG scores at baseline would act as a buffer against symptom increase in response to revictimization.

## METHODS

### PROCEDURE AND PARTICIPANTS

Participants were recruited through the Dutch Victim Compensation Fund (DVCF) and were victims of different types of interpersonal violence who had claimed for financial compensation with the DVCF between January 1st and December 31st, 2006. All had taken part in an earlier cross-sectional PTSD study conducted in October-December 2007 (T1;  $n = 744$ ) and had provided informed consent to participate in a follow-up study (Kunst, Winkel, & Bogaerts, in press). File inspection suggested that 34 (16.8%) of them could be broadly classified as victims of sexual assault, 68 (33.7%) as victims of minor physical assault, and 55 (27.2%) as victims of robbery, according to the legal descriptions of the events they had experienced. Forty-five (22.3%) participants had experienced another type of violence.

Potential participants were approached with a letter that explained the purpose of the study and invited to fill out a set of questionnaires on PTSD and violent revictimization six months after the cross-sectional study (T2). The survey was to be completed online, yet those who did not have access to the World Wide Web or preferred to fill out the questionnaire on paper could request for a hardcopy version. Of the 640 victims who had agreed to participate in the current study, 235 responded (36.7%). Approval for the study was obtained from the DVCF Committee.

Only respondents without missing values were included in statistical analyses. Missing data on study variables were not estimated using statistical imputation procedures, since the program running the questionnaires did not allow participants to skip a particular question and continue with the next question. Consequently, the pattern of missingness for successive questions could not be investigated: 208 had no missing values at T1 and T2. Six participants were excluded due to outliers. Thus, the final sample contained 202 individuals. Participants included in statistical analyses were slightly older than nonresponders and those who were excluded from analytic procedures ( $p < .05$ ). No differences between responders and nonresponders were found on T1 measurements of PTSD, PTG, and any of the remaining background variables.

As the author availed of information on age, gender, and time since victimization of all victims who had filed a claim with the DCVF in 2006 and had been eligible for participation in the first study (i.e., no missing data on any of these background variables, as this would indicate that the victim involved had failed to return the application form after an initial request for sending a copy through the mail;  $n = 3721$ ; see Kunst et al., in press), the representativeness of the study sample with regard to these background variables could be assessed. Participants were more often female (61.9% versus 48.6%,  $\chi^2 = 13.55$ ,  $p < .001$ ) and older ( $M = 44.23$ ,  $SD = 15.90$  versus  $M = 39.30$ ,  $SD = 15.53$ ,  $p < .001$ ) than applicants who had not participated. No differences between participants and nonparticipating victims were observed for time since victimization ( $M = 4.77$  years,  $SD = 3.56$  versus 5.11 years,  $SD = 4.28$ ,  $p = ns$ ).

## MEASURES

*Revictimization.* Revictimization was assessed using three items regarding different types of violence (sexual harassment, threat of physical violence, and actual physical violence). Previous research suggests that comparable short screeners of recent victimization experiences are highly accurate classification instruments (e.g., Norris & Kaniasty, 1994). Items were derived from the Dutch Safety Survey (DSM) 2008 (Centraal Bureau voor de Statistiek, 2008) and covered all the broad categories of violent crime assessed by this questionnaire. Participants were allowed to report more than one victimization experience. In total, 20 participants reported being sexually



harassed during follow-up, 19 had been threatened with violence, and 7 had experienced physical violence.

*Posttraumatic Growth Inventory.* Perceptions of PTG were measured by the Dutch version of the Posttraumatic Growth Inventory (PTG; Jaarsma, Pool, Sanderman, & Ranchor, 2006). The PTGI was originally developed by Tedeschi and Calhoun (1996) and taps five domains of PTG: relating to others, new possibilities, personal strength, spiritual change, and appreciation of life. Each item needs to be answered on a 6-point Likert scale (0 = not at all, 5 = extremely). Both subscale and total scale scores can be calculated. For the current study, only the PTG total score was used. Participants were instructed to report PTG levels related to the act of violence leading to the request for compensation. The psychometric properties of the PTGI have been well established in victims of violence (e.g., Crubaugh & Resick, 2007; Cobb, Tedeschi, Calhoun, & Cann, 2006; Updegraff & Marshall, 2005). Internal consistency reliability for the PTG in the present study was  $\alpha = .95$ .

*PTSD Symptom Scale, Self-Report Version.* PTSD symptom severity was mapped with the PTSD Symptom Scale, Self-Report version (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993). Its 17 items correspond to the PTSD symptoms listed in the Diagnostic and Statistical Manual of Mental disorders, third edition (DSM-III; 1987). The PSS-SR has often been used as a screening instrument for PTSD symptomatology among victims of crime (e.g., Dunmore, Clark, & Ehlers, 1999; Rose, Brewin, Andrews, & Kirk, 1999; Andrews, Brewin, Rose, & Kirk, 2000). The Dutch version of the PSS-SR has been constructed by Arntz (1993). Recently, several studies have used web-based versions of the PSS-SR (e.g., Elhai & Simons, 2007). For each of the 17 items, respondents had to indicate to what extent they had experienced the corresponding symptom during the past week on a 4-point Likert scale (0 = never, 1 = once, 2 = 2–4 times, 3 = 5 times or more). PSS-SR total scale as well as subscale scores for intrusion, avoidance, and hyperarousal symptoms can be computed by summing item scores, although for the current study only PSS-SR total scores were used. Both at T1 and T2, participants were instructed to fill out the PSS-SR with regard to the violent incident that had resulted in application for compensation from the fund. The psychometric properties of the PSS-SR have been found to be satisfactory in samples containing victims of violence (Wohlfarth, Van den Brink, Winkel, & Ter Smitten, 2003; Foa et al., 1993). In the



current study, the PSS-SR demonstrated good internal consistency reliabilities, with  $\alpha = .94$  at T1 and  $.92$  at T2.

## STATISTICAL ANALYSES

All statistical analyses were performed using the software package SPSS 15.0 for Windows (SPSS Inc., Chicago, Illinois). As a preliminary step, revictimization scores were dichotomized (revictimization = 1) prior to inclusion in subsequent analyses; this was deemed appropriate as only eight of those revictimized ( $n = 35$ ) had experienced more than one act of violence during the follow-up. To provide a description of the study sample, means, standard deviations, and bivariate correlations were calculated for each variable.

Next, a one-way ANCOVA was carried out to test whether revictimization was associated with PTSD symptom severity at T2, while controlling for PTSD symptom severity at T1.

Subsequently, in accordance with statistical procedures prescribed to test for moderation (Cleary & Kessler, 1982; Cohen & Cohen, 1983; Baron & Kenny, 1986), a stepwise regression analysis was carried out to investigate whether PTG moderated the association between revictimization and PTSD symptom severity at follow-up. PTSD symptom severity at T1, PTG, and potential covariates (i.e., age, gender, and time since victimization) were entered on step 1, while the interaction term for revictimization and PTG was added in step 2. To test whether any of the covariates interacted with the two independent variables, the model was extended with a final step that included all interactions between covariates and independent variables. All continuous predictors were centered prior to entry in the model to avoid multicollinearity (cf. Holmbeck, 2002, p. 89). Residual analysis did not indicate violation of assumptions underlying multiple regression analysis (i.e., normality, linearity, and homoscedasticity between predicted dependent variable scores and errors of prediction).

If the interaction between revictimization and PTG at T1 proved to be significant, simple regression lines for high and low values of the moderator variables were plotted to visually inspect the manner in which revictimization and PTG interacted. To obtain accurate figures, we followed a slightly modified version of the *Post Hoc Probing of Significant Moderational Effects* procedure described by Holmbeck (1997, 2002), which draws from the work provided by Cohen

and Cohen (1983) and Aiken and West (1991). First, two conditional moderator variables were created for respondents scoring high (1 SD above the centered sample mean) and low on PTG (1 SD below the centered sample mean), respectively. Second, using the entire study sample, two separate regression analyses were run to generate simple slopes for high and low PTGs and to determine their significance in predicting PTSD symptom severity (PSS). They were conducted by simultaneously entering the main effect for revictimization, one of the conditional moderator variables, the interaction between the categorical revictimization variable and one of the conditional moderator variables, and covariates. Finally, the regression lines were plotted by substituting revictimization scores (0 or 1) in the generated equations and adding the constant for each equation.<sup>1</sup> The constant was calculated by summing the constant provided by the SPSS output and the products of the sample means of all covariates and their regression weights (Holmbeck, 2002).

## RESULTS

Descriptive statistics are presented in Table 1. Although a significant correlation existed between revictimization and PTSD symptom severity at T2, ANCOVA revealed no main effect for revictimization on PTSD symptom severity at T2 when adjusting for the influence of PTSD symptom severity at T1. The results of the stepwise regression analysis are presented in Table 2. Neither revictimization nor PTG were significantly associated with PTSD symptom severity at T2 on step one. PTSD symptom severity at T1 was the only significant predictor of PTSD symptom severity at T2 in this step. However, step 2 revealed that the interaction between revictimization and PTG significantly predicted PTSD symptom severity at T2

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1. This part of our procedure differs from the computational examples provided by Holmbeck (2002). In his 2002 paper, Holmbeck provides, among other things, two computational examples of post-hoc probing: one involving a two-way interaction between one dichotomous variable (the moderator) and one continuous variable (the independent variable to be moderated) and one involving two continuous variables. In both cases regression lines can be plotted by first centering the independent variable and then substituting high (1 SD above the mean) and low (1 SD below the mean) values of the centered independent variable. However, as the revictimization variable (the independent variable of interest in our model) was dichotomous in nature, centering was not necessary to create two separate conditions; instead, values for the independent variable can be substituted straightway (G.N. Holmbeck, personal communication, April 3, 2009),

TABLE 1: Sample Means, Standard Deviations, and Pearson Correlation Coefficients

Variables	M	SD	1	2	3	4	5	6	7
1. Age	44.23	15.90	1.00						
2. Sex (female)	.62	.49	-.16*	1.00					
3. Time since victimization	5.11	4.28	-.14	.12	1.00				
4. PTSD symptom severity at T1	15.70	12.75	.11	.14	.19**	1.00			
5. Revictimization†	.17	.38	-.11	.12	.13	.18*	1.00		
6. PTG at T1	40.58	25.68	-.18*	.22**	.11	.11	.05	1.00	
7. PTSD symptom severity at T2	13.31	10.77	.08	.16*	.18*	.73***	.18*	.01	1.00

†Note that revictimization was coded as revictimization = 1. PTG refers to posttraumatic growth. \* $p < .05$ ; \*\* $p < .01$ ; \*\*\* $p < .001$ .

TABLE 2. Stepwise Regression Analyses Predicting PTSD Symptom Severity at T2

Variable	B	SE B	$\beta$	$R^2$
Step 1				.553***
Age	.01	.03	.01	
Sex (female)		1.55	1.11	.07
Time since victimization	.10	.13	.04	
PTSD symptom severity at T1	.61	.04	.72***	
Revictimization	1.26	1.40	.04	
PTG at T1	-.04	.02	-.09	
Step 2				.563***
Age	.00	.03	.00	
Sex (female)	1.36	1.10	.06	
Time since victimization	.10	.12	.04	
PTSD symptom severity at T1	.60	.04	.71***	
Revictimization	1.58	1.40	.06	
PTG at T1	-.02	.02	-.05	
Revictimization X PTG at T1	-.12	.06	-.11*	

Note that PTG refers to posttraumatic growth. \* $p < .05$ ; \*\*\* $p < .001$ .

above and beyond baseline symptom levels. Step 3 did not significantly increase the explanatory power of the model and was therefore dropped from the model (cf. Holmbeck, 2002; Frazier, Baron, & Tix, 2004).

Post hoc probing revealed the following regression equation for victims with high levels of PTG:  $PSS = 22.53 - 1.44$  (revictimization). The simple slope of this equation (i.e., the coefficient for the revictimization variable) was not significant. In contrast, a significant slope was obtained for low PTGs,  $t(201) = 2.17$ ,  $p < .05$ . The regression equation for this group was:  $23.56 + 4.61$  (revictimization). The graphical depiction of these results clearly supports the moderation hypothesis proposed in the introduction. Victims with low levels of PTG appear to be at risk of experiencing an increase in PTSD symptom severity in response to revictimization. On the other hand, follow-up symptom levels for high PTGs do not seem to depend on revictimization status (see Figure 1).

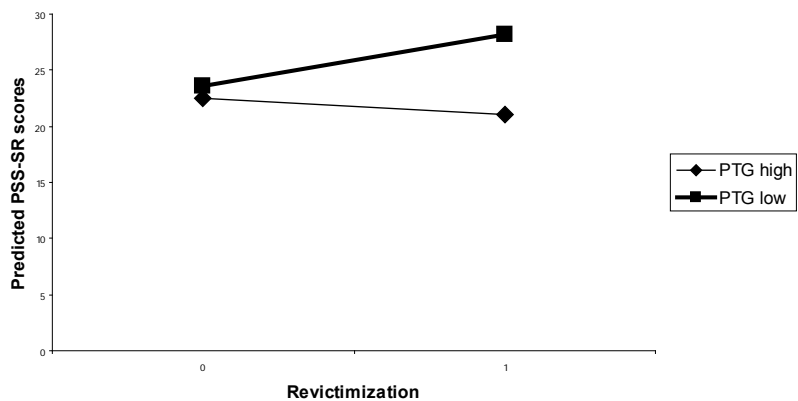


FIGURE 1. Relationship between revictimization (yes = 1, no = 0) and PTSD symptom severity at T2 for participants scoring low (1 SD below the centered mean) and high (1 SD above the centered mean) on posttraumatic growth (PTG)

DISCUSSION

The present study was developed to assess the protective potential of PTG among victims of violence in dealing with subsequent violence exposure. Contrary to expectations, revictimization was not associated with PTSD symptom severity at T2 in response to the original event above and beyond PTSD symptom levels at T1. This may be due to the prospective design of our study, which allowed for adjustment of PTSD symptom severity prior to revictimization. Most previous studies that investigated the adverse effect of revictimization were not able to do so and therefore may have found biased results; parameter biases may occur when relevant variables are not taken into account in statistical analyses (e.g., Tabachnick & Fidell, 2007). Another explanation is that the reported acts of revictimization were not severe enough to have an independent impact on PTSD symptom severity. Unfortunately, as we did not assess the perceived impact of revictimization, we were not able to check this. On the other hand, it is worth noting that the failure to find an independent effect for violent revictimization seems to be in line with a recent prospective study by Breslau, Peterson, and Schultz (2008), who found that prior trauma increases the risk of PTSD after a sub-

sequent trauma only among persons who had developed PTSD in response to the prior trauma.

For PTG a main effect was not found either. This finding fits well with the abundance of studies that have failed to find a (strong) relationship between PTG and adverse outcomes (Zoellner & Maercker, 2006). However, and as expected, an interaction effect was observed between revictimization and PTG. Post hoc probing suggested that victims scoring low on PTG developed increased levels of PTSD symptom severity after reexperiencing an act of violence.

Although preliminary in nature, our findings may prove to be of importance for those working with victims of violence, since they suggest that PTG buffers against symptom increase after revictimization and thus prevents disruption of the coping process initiated by the initial act of violence. The buffering effect of PTG is even more compelling as one considers that (1) victims of violence are at an increased risk of being victimized again compared to nonvictims and (2) that the mean sample score for PTSD symptom severity was above the cut-off ( $\geq 15$ ) established by Wohlfarth, Van den Brink, Winkel, and Ter Smitten (2003) to classify subjects with PTSD diagnosis. For some individuals, promoting PTG among those who have not experienced PTG by themselves might prove to be effective in counterbalancing the negative cumulative impact of subsequent adverse events, such as revictimization. How PTG should be promoted in victims of violence remains subject for future research though. To date, many scholars active in the field of PTG have merely stressed the importance of enhancing PTG without indicating which strategies should be considered to accomplish this and how they should be adapted to the needs of specific subgroups, such as those who consider themselves unable to perceive PTG (e.g., Woodward & Joseph, 2003; Frazier & Berman, 2008). Except from several writing studies, which failed to find convincing support for writing instructions to enhance levels of PTG (e.g., Weinrib, Rothrock, Johnsen, & Lutgendorf, 2006; Gebler & Maercker, 2007) or did not check whether the intervention actually facilitated an increase in PTG (e.g., McCullough, Root, & Cohen, 2006), most clinical interventions appear not to have been designed to increase PTG (Park & Helgeson, 2006). Important to note in this respect is that factors suggested to be associated with the concept of PTG in the context of violence, such as forgiveness (e.g., Wright, Crawford, & Sebastian, 2007; Fisher, 2006; Tedeschi, 1999), do not necessarily provide pathways to induce or

increase PTG. Until further insight has been gained in the potential utility and effectiveness of interventions aimed at fostering PTG and their associations with PTSD, caution should be taken when applying them to victims of violence seeking professional help. Inspired by the current study, future studies may consider exploring the impact of instructions to write about PTG which are embedded in vignettes that point to the risk of revictimization. By encouraging victims to actively reflect on positive outcomes of the initial act of violence, a backdrop in psychological functioning might be prevented when revictimization actually occurs.

As an alternate explanation for our findings, one might argue that it is not so much the experience of PTG itself that protects traumatized individuals against symptom increase resulting from revictimization after initial traumatization yet merely the psychological resources that enabled them to experience PTG in the first place (e.g., Tennen & Affleck, 1998), particularly global personality traits, such as optimism and positive affectivity. Furthermore, among those experiencing PTG, such resources may determine which persons are most likely to profit from PTG in an adaptive manner (e.g., Erbes, Eberly, Dikel, Johnsen, Harris & Brigdahl, 2005; Park & Fenster, 2004). Similarly, Milam (2006) posited that PTG reflects an active coping approach resulting in beneficial outcomes in people with positive expectations about the future, whereas maladaptive outcomes may be observed in those who employ PTG as an avoidant-denial strategy stemming from underlying negative expectancies. Previously, these contradicting sides of PTG have been qualified as "the Janus-Face of PTG" (Maercker & Zoellner, 2004; Zoellner & Maercker, 2006). Preliminary support for these suppositions was found by Updegraff and Marshall (2005). They examined the predictive value of dispositional optimism on PTG in victims of community violence and found that optimism assessed several days after hospital admission was associated with PTG at three months follow-up.

## STUDY LIMITATIONS AND STRENGTHS

Despite the promising results of our study, several limitations need to be mentioned as well. First, our sample consisted of victims of violence applying for compensation from the state. It is possible that our findings do not apply to victims of violence not seeking



financial support with government bodies. Second, the rather low response rate may indicate that our sample was not representative of the entire population of victims applying with the fund. Analyses testing differences between participants included in our final sample and those who did not participate partly supported this hypothesis. Third, as mentioned above, we did not assess the perceived severity of the acts of revictimization and therefore were not able to determine to what extent they were actually stressful to those revictimized. Finally, within-participants differences on time since revictimization may have influenced the reliability of our results. These limitations can not refute, however, that the current study was the first to investigate the adaptive potential of PTG among victims of violence in the face of revictimization and did so by using a prospective design.

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